

# Aquatic Toxicity of Nine Aircraft Deicer and Anti-Icer Formulations and Relative Toxicity of Additive Package Ingredients Alkylphenol Ethoxylates and 4,5-Methyl-1H-benzotriazoles<sup>†</sup>

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Characterization of the effects of aircraft deicer and anti-icer fluid (ADAF) runoff on aquatic organisms in receiving streams is a complex issue because the identities of numerous toxic additives are proprietary and not publicly available. Most potentially toxic and endocrine disrupting effects caused by ADAF are due to the numerous additive package ingredients which vary among manufacturers and types of ADAF formulation. Toxicity investigations of nine ADAF formulations indicate that endpoint concentrations for formulations of different manufacturers are widely variable. Type IV ADAF (anti-icers) are more toxic than Type I (deicers) for the four organisms tested (*Vibrio fischeri*, *Pimephales promelas*, *Ceriodaphnia dubia*, and *Selenastrum capricornutum*). Acute toxicity endpoint concentrations ranged from 347 to 7700 mg/L as ADAF for Type IV and from 1550 to 45 100 mg/L for Type I formulations. Chronic endpoint concentrations ranged from 70 to 1300 mg/L for Type IV and from 37 to 18 400 mg/L for Type I formulations. Alkylphenol ethoxylates and tolyltriazoles are two known classes of additives. Nonylphenol, nonylphenol ethoxylates, octylphenol, octylphenol ethoxylates, and 4,5-methyl-1H-benzotriazoles were quantified in the nine ADAF formulations, and toxicity tests were conducted with nonylphenol ethoxylates and 4,5-methyl-1H-benzotriazoles. Toxicity units computed for glycol and these additives, with respect to toxicity of the ADAF formulations, indicate that a portion of ADAF toxicity can be explained by the known additives and glycols, but much of the toxicity is due to unidentified additives.

## Introduction

Releases of aircraft deicers and anti-icers (ADAFs) to the environment have potential to cause damaging effects to

aquatic ecosystems including aquatic toxicity (1–3), depressed dissolved oxygen due to elevated biochemical oxygen demand (BOD) (4–6), and possible endocrine disruption due to degradation products of ADAF additives (7). The U.S. EPA has estimated that 40 million liters of ADAF are currently discharged to surface waters each year (8). In a survey of European airports, numerous airports reported that no attempt was made to recover spent ADAF (9).

ADAFs are commonly applied to the wings and fuselage of aircraft during cold weather to remove and prevent snow and ice buildup on aircraft surfaces that could otherwise impede safe air travel. Many airports located in colder climates use ADAF nearly every day of the winter season to some degree. Airports in warmer climates use them less frequently, but freezing precipitation warrants the use of ADAF nearly every year even at some warmer climate airports such as those in the southern United States. During ADAF application operations, airports and airlines are faced with a formidable combination of tasks including the removal of ice and snow from aircraft surfaces, the prevention of ice and snow accumulation on aircraft surfaces before takeoff, maintaining ice- and snow-free taxiways and runways, maintaining flight schedules, and minimizing environmental impact of ADAF through deicer management.

The freezing-point depressants in ADAF are typically propylene glycol (PG), ethylene glycol (EG), or diethylene glycol. ADAF also contains water and various additives, collectively referred to as the additive package, which serves to enhance performance of ADAF. Fluid designations are based on aerospace material specifications published by the Society of Automotive Engineers (SAE). Type I fluids are deicers used for removing ice, frost, and snow from aircraft surfaces. Type I fluids are diluted with as much as 80% water and heated to between 150 and 180 °C before application. Type II and IV fluids are more viscous anti-icers applied full strength at ambient temperatures to prevent the formation of ice and snow on aircraft (5). Type IV anti-icers are more commonly used by major airlines, while Type II anti-icers are mostly used by smaller airlines due to financial considerations. Other classes of chemicals in additive packages include corrosion inhibitors, surfactants, thickeners, dyes, flame retardants, and pH buffers (5).

The fate of ADAF varies depending on the individual airport, the deicer collection facilities implemented, and the nature and timing of precipitation. Overspray during initial application, dripping from the aircraft during holdover activities (the wait between ADAF application and takeoff), shear during takeoff, and melting from accumulated ADAF in snowbanks can lead to ADAF releases to surface water and groundwater. Many airports have implemented some form of ADAF management to reduce runoff to receiving waters. Included in these management practices are containment measures such as deicing pads, glycol recovery vehicles, storm sewer balloons, and snow containment systems (10). Some airports also choose to reduce ADAF usage at the source by variably mixing water with ADAF (10), mixing with forced hot air during application, or using nonglycol alternatives such as infrared technology to remove ice and snow from the aircraft.

ADAF additives rather than glycol have been implicated as the primary source of ADAF toxicity (1, 2, 11, 12), but manufacturers of ADAF maintain the proprietary nature of additive package formulas and are not required to reveal the contaminants responsible for toxicity. Of the many additives in ADAF, researchers have identified two classes of chemicals that are of concern with regard to aquatic toxicity. The first

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class includes benzotriazole and benzotriazole derivatives that are used as corrosion inhibitors in some ADAP formulations (13, 14). The two isomers 4- and 5-1H-methylbenzotriazole (MeBT) are thought to be the benzotriazole derivatives of greatest concern with regard to ADAP runoff (14, 15). These additives are a source of toxicity to microorganisms (13), and have been detected in water receiving airport runoff at concentrations of toxicological significance (14, 16, 17). MeBT, however, is not the only source of toxicity in ADAP; other additive package components are responsible as well (15).

The second class of identified additives includes alkylphenol ethoxylate (APEO) surfactants. These surfactants were identified in five of nine ADAP formulations previously tested (7) including nonylphenol ethoxylates (NPEO) and octylphenol ethoxylates (OPEO). The products used in commerce are almost exclusively oligomeric mixtures of ethoxy-substituted phenyl compounds. The alkyl group, typically in *para*-position with respect to the phenyl, determines whether the surfactant is NPEO (containing nine carbons with multiple branching) or OPEO (containing eight carbons that have a symmetrical 1,1,3,3-tetramethylbutyl structure). Products with NPEOs typically contain average ethoxylate chain lengths between 4 and 20 carbons, while selected products can have average chain lengths as high as 100 and as low as 0 (18). These surfactants are of special concern with regard to aquatic toxicity due not only to toxicity of the parent products, but the potential of these compounds to become more toxic through the degradation process. The degradation products nonylphenol (NP), octylphenol (OP), the smaller chain ethoxomers ( $n = 1$  and 2), and alkylphenol ether carboxylates are more toxic than the larger chain ethoxomers (19). Some of the degradation products are also endocrine disruptors including NP, OP, and nonylphenol ether carboxylates (NPnEC,  $n = 0,1$ ) (20, 21).

Other reported ADAP components that have not been studied as thoroughly with regard to ADAP runoff include alcohol ethoxylate surfactants such as lauryl alcohol, decyl alcohol, and lauryl alcohol-phosphoric acid ester (7) as well as diethylene glycol, ethylene oxide, acetaldehyde, dioxane, high-molecular-weight polymers, polyamines, ureas, sodium nitrate, sodium benzoate, and Borax (5, 22).

Glycol concentrations in U.S. and Canadian airport runoff have been detected in the thousands of mg/L, and at times, greater than 100 000 mg/L (1, 2, 6, 17, 23, 24). Currently, freshwater guidelines for EG and PG in Canada are 192 and 500 mg/L, respectively. ADAP runoff is presently regulated by individual states in the United States, but the U.S. EPA is currently studying ADAP runoff in consideration of national regulation alternatives. Though regulation of ADAP runoff in the United States is not typically directed toward toxicity endpoints, SAE has revised the Aerospace Material Specification for Type I ADAP to include aquatic toxicity standards (25). These standards apply to the approval of new Type I formulations entering the market, but not formulations that have been previously approved by SAE. Type IV fluids, the more toxic of the two types of ADAP, do not have SAE aquatic toxicity standards. The standards specify a limit of 4000 mg/L for LC<sub>50</sub> endpoint concentrations (the concentration at which there is 50% mortality) computed as ADAP for fathead minnows, *Ceriodaphnia dubia*, *Daphnia magna*, and rainbow trout. While these standards are a step in the right direction, it is apparent that U.S. airports discharge ADAP at concentrations greater than 4000 mg/L at times. Even at levels less than 4000 mg/L, sublethal toxic effects will be exerted on aquatic organisms.

The overall objective of this ongoing research is to study the effects of ADAP runoff on receiving water. Specific objectives of this paper were to describe toxicity of different Type I and Type IV formulations, describe the content of

**TABLE 1. Freezing Point Depressant and Toxicity Test Dates for Five Type I Aircraft Deicing Fluids and Four Type IV Aircraft Anti-Icing Fluids from Four Different Manufacturers**

manufacturer	fluid type	short name	freezing point depressant	test dates
1	I	Type I-1a	PG	Apr 1997–Aug 2003
1	I	Type I-1b	PG	May–July 2005
2	I	Type I-2	PG	Feb 2003
3	I	Type I-3	PG	Apr–June 2004
4	I	Type I-4	EG	May–June 2003
1	IV	Type IV-1	PG	Feb–Mar 2003
2	IV	Type IV-2	PG	Mar–Jun 2003
3	IV	Type IV-3	PG	Apr–June 2004
4	IV	Type IV-4	EG	Feb–June 2003

MeBT and APEO in these formulations, and define the toxicity due to glycols, MeBT, and APEO as opposed to other additives in ADAP formulations.

## Materials and Methods

**ADAP Collection.** Undiluted ADAP formulations were collected directly from storage tanks and deicing/anti-icing vehicles from General Mitchell International Airport in Milwaukee, WI (PG formulations) and Dallas Fort Worth International Airport (EG formulations). Tests on formulations were conducted within two months of sample collection.

**Toxicity Tests.** Toxicity tests were conducted on five Type I ADAP formulations and four Type IV ADAP formulations between April 1997 and July 2005. Four of the Type I formulations were PG based and the fifth was EG based. Three of the Type IV formulations were PG based and the fourth was EG based (Table 1). Toxicity tests were also conducted on 5 MeBT, a mix of 4,5 MeBT, and an NPnEO mixture ( $n = 1–17$ ) with a maximum ethoxomer concentration at  $n = 10$  and a mean at  $n = 9.4$  (table A1, Supporting Information). This NPnEO mixture includes a commercial surfactant (Huntsman Surfonic N-95) with NP and NPnEO ethoxomers with  $n = 1$  and 2 added to approximate the distribution of NPnEO in ADAP formulation Type I-1a. This was the ADAP formulation used most extensively at General Mitchell International Airport through spring of 2005. Toxicity endpoint concentrations for PG and EG are also presented; of which, the Microtox values were generated during this research and the *P. promelas*, *C. dubia*, and *S. capricornutum* values were taken from previously published research (12, 26). When ADAP samples were not in use, the products were stored in a dark walk-in cooler at 4 °C. Initial range-finding tests were conducted to approximate the appropriate toxic concentration for each test organism. ADAP concentrations are reported as nominal (concentrations were calculated from dilutions). A 0.5 dilution series was prepared for these tests with hard-reconstituted water (hardness of 180 mg/L as CaCO<sub>3</sub>) for dilution, resulting in five ADAP treatments and a control for each test. All toxicity tests met control criteria established by the U.S. EPA for whole effluent toxicity tests. Toxicity tests were conducted at the Wisconsin State Laboratory of Hygiene in Madison, Wisconsin.

**Microtox Assays.** Microtox assays were conducted to determine the EC<sub>50</sub> for the marine bacterium *Vibrio fischeri* using freeze-dried bacteria supplied by Azur Environmental of Carlsbad, CA. *V. fischeri* were rehydrated in a reconstitution solution that was also supplied by Azur Environmental. Sodium chloride (Fisher Scientific, Pittsburgh, PA) was added to each treatment to provide a 2% saline environment. Test temperatures were 15 °C. The bacteria emitted light when healthy; stress was displayed by suppressed light emission. Light emission was recorded at 5 and 15 min. The percent

effect ((light lost/lab control value)  $\times$  100) of the 15 min light reading was used as the endpoint. Adverse effects to the test bacterium were indicated when the percent effect was greater than zero.

**Acute-Toxicity Tests.** Acute toxicity tests were conducted using juvenile *Pimphales promelas* (4–10 days old, 10 replicates) and *Ceriodaphnia dubia* (<24 h old, four replicates). Acute toxicity tests were conducted following standard U.S. EPA methods (27). All 48 h *C. dubia* and 96 h *P. promelas* tests were conducted at 20 °C with a 16:8-hour light/dark cycle. Treatments were prepared each day for renewal of test water. During the acute tests, *C. dubia* were not fed and *P. promelas* were fed once at 2 h prior to the 48 h renewal. Initial and final temperature, DO, pH, and conductivity were measured daily. Survival was recorded for each treatment at the termination of each test.

**Chronic Toxicity Tests.** *P. promelas*, *C. dubia*, and the green alga, *Selenastrum capricornutum*, were also tested in accordance with standard U.S. EPA methods (28) to determine chronic effect concentrations (sublethal endpoints) for ADAF. Age of organisms at test initiation were <24 h for *P. promelas* and *C. dubia* (*C. dubia* were all within an 8 h age range) while algae were harvested from a 4–7 day old culture in log phase growth. Five, ten, and four replicates were used in *P. promelas*, *C. dubia*, and *S. capricornutum* chronic tests, respectively. Static renewal chronic animal tests were done at a temperature of 25 °C with a 16:8-hour light/dark cycle. Chronic test animals were fed throughout the exposure period. Live brine shrimp were fed to the fish three times daily. The *C. dubia* chronic test organisms were fed a combination of yeast/cerophyll/trout food and green algae (*S. capricornutum*). The fish tests were terminated on day 7, when the fish were sacrificed, dried, and weighed for determination of growth which was the chronic endpoint. *C. dubia* chronic tests were terminated after 80% of controls released their third brood (6–7 days). The total number of young produced per original female was used as the chronic endpoint.

Chronic tests with the green alga, *S. capricornutum*, were conducted following previously published modifications (29) to the U.S. EPA algal test method (28). Algal growth nutrients were added to the samples. Test chambers were 48 well microplates (Falcon, Lincoln Park, NJ) with 1 mL sample aliquots in four replicate wells. Approximately 10 000 algal cells were added to each well. Under continuous 4000 lux lighting, microplates were covered and placed on a shaker table inside a 25 °C incubator for 96 h. To account for light and temperature variations, the shaker table was rotated 90° every 24 h. Growth was measured fluorometrically and used as the chronic endpoint.

Microtox software was used to calculate the EC<sub>50</sub> values for all 15 min readings (30). LC<sub>50</sub> values for acute tests were computed using the probit or trimmed Spearman–Kärber method, as appropriate (27). The IC<sub>25</sub> for each chronic test was computed using the IC<sub>p</sub> method developed by U.S. EPA (31).

**Chemical Analysis.** *Alkylphenols and Alkylphenol Ethoxylates.* The different ADAF formulations were analyzed for NP, octylphenol (OP), nonylphenol ethoxylates (NP1–16EO), and octylphenol ethoxylates (OP1–5EO). Approximately 1 g of each formulation was weighed and dissolved in 10 mL of a 50:50 methanol/water mixture. Methanol was acquired from Burdick & Jackson (Honeywell International Inc., Muskegon, MI), and water was organic-free deionized (18.2 MΩ-cm) water obtained from a NANOpure system (Barnstead International, Dubuque, IA). Aliquots of 1.5 mL from the resulting 100 mg/mL solutions were transferred to vials and spiked with a mixture of <sup>13</sup>C<sub>6</sub>-labeled internal standards. These were then injected into a Waters 2690 XE separations module (Waters Corp., Milford, MA) with a 4.6  $\times$  150 mm MSPak GF-310 4D column (Shodex, Shoko Co., Tokyo, Japan)

interfaced with a Quattro LC triple quadrupole mass spectrometer (Micromass Ltd., Manchester, UK) with an electrospray ionization source. Specific ions analyzed, MS conditions, and details of the quantitation method were reported elsewhere (32, 33). Resulting detection limits ranged from 11 (NP16EO) to 221 ng/g (OP).

**4,5-Methyl-1H-Benzotriazole.** A 10 mL aliquot of each ADAF was weighed and diluted to 100 mL with methanol (Burdick & Jackson, Muskegon, MI). Each diluted ADAF was injected into a gas chromatograph with a flame ionization detector. Concentrations were calculated by comparing responses to a five point calibration curve using 5-MeBT (Aldrich Chemical, Milwaukee, WI) and 4-MeBT (University of Colorado, Boulder, CO). The resulting detection limit was 100 µg/g for both isomers.

**Data Analysis.** Toxicity units (TU) were computed to compare additive toxicity to overall ADAF formulation toxicity using the following formula: TU = (fraction of additive in ADAF  $\times$  endpoint concentration for ADAF)/(endpoint concentration for additive). The resulting TU describes the fraction of overall ADAF toxicity that is explained by the selected additive. For example, a TU of 0.5 for additive<sub>a</sub> with respect to ADAF<sub>a</sub> indicates that the concentration of additive<sub>a</sub> would be <sup>1</sup>/<sub>2</sub> of the LC<sub>50</sub> for additive<sub>a</sub> in a dilution concentration of ADAF<sub>a</sub> equal to the LC<sub>50</sub> of ADAF<sub>a</sub>.

## Results and Discussion

**Toxicity of ADAF Formulations.** Tested organisms were shown to be more sensitive to Type IV formulations than Type I formulations (Table 2). Additionally, toxicity endpoints varied depending on the formulation tested within the Type I formulations and within the Type IV formulations. Acute toxicity endpoints for Type I formulations ranged from the 1550 mg/L for formulation Type I-1a to 45 100 mg/L for formulation Type I-1b. Acute toxicity endpoints for Type IV fluids were much lower with all values less than 2000 mg/L except the Microtox EC50s for formulations Type IV-2 and Type IV-4.

Of the Type I fluids, all species except *S. capricornutum* were most sensitive to formulation Type I-1a while all species except *S. capricornutum* were least sensitive to formulation Type I-1b. Of the Type IV fluids, all species were most sensitive to either formulation Type IV-3 or Type IV-4 except *S. capricornutum*.

Consistent with previous studies, these ADAF formulations exhibited greater toxicity than pure EG or PG confirming that components of the various additive packages were responsible for much of the observed toxicity (12, 23, 26). Since the additive package formulations are proprietary, most of the additives are not publicly known; however, two classes of additives that have been identified were explored further.

**APEO and MeBT content in ADAF.** APEO surfactants and MeBT were quantified in each of the nine ADAF formulations used for this study (Table 3). APEO was detected in six of the nine formulations with only NPEO in two of the formulations and both NPEO and OPEO in four of the formulations. Three of the formulations (Type I-1a, Type IV-2, and Type IV-4) stand out as having greater APEO content than others, and Type IV-4 has more than three times the APEO than any other formulation. It should also be noted that OPnEO for *n* > 5 have not been quantified, but analysis confirms that all of the formulations with detected OPEO have ethoxomers present beyond the 5th ethoxomer at least as high as *n* = 15. For this reason, total OPnEO content in Table 3 is underestimated.

NPEO maximum individual ethoxomer concentrations for different ADAF formulations varied between the 3rd and 11th ethoxomer, while the mean ethoxomer numbers varied between 4.8 and 10.8 depending on the ADAF formulation. It was not possible to determine maximum or mean OPEO

**TABLE 2. Acute and Chronic Toxicity Test Results for Five Type I Aircraft Deicing Fluids and Four Type IV Aircraft Anti-Icing Fluids from Four Different Manufacturers (All Units Are mg/L of Neat ADAF)**

formulation	specific gravity	% glycol	acute			chronic		
			Microtox (EC50)	<i>P. promelas</i> (LC50)	<i>C. dubia</i> (LC50)	<i>P. promelas</i> (IC25)	<i>C. dubia</i> (IC25)	<i>S. capricornutum</i> (IC25)
Type I								
Type I-1a	1.04	88	1,550 (1290–1760)	1,910 (1,700–2,100)	3,380 (2,390–4,780)	1,600 (1,290–1,800)	567 (437–1028)	242 (225–254)
Type I-1b	1.04	88	44,500 (37,000–50,200)	30,800 (27,000–35,200)	45,100 (38,900–52,500)	18,400 (16,100–20,000)	6,390 (5,500–7,340)	37.0 (28.0–51.0)
Type I-2	1.04	88	5,270 (4,810–5,880)	6,740 (5,980–7,590)	5,970 (5,000–7,100)	8,530 (5,730–10,000)	2,920 (2,220–3,380)	14.2 (11.8–16.8)
Type I-3	1.04	88	14,400 (14,000–15,000)	12,300 (10,800–14,100)	7,850 (6,810–9,040)	6,060 (5,360–6,260)	2,860 (2,660–2,950)	332 (146–429)
Type I-4 <sup>a</sup>	1.11	92	11,900 (11,200–12,500)	24,700 (21,700–28,100)	15,700 (13,700–18,100)	4,430 (3,420–7,460)	5,470 (2,060–6,440)	4,550 (3,290–5,580)
Type IV								
Type IV-1	1.06	>50	663 (622–708)	1,690 (1,370–2,100)	575 (458–709)	1,300 (997–1,500)	332 (96.8–425)	34.3 (33.2–35.4)
Type IV-2	1.04	>50	4,550 (4,310–4,880)	932 (863–1,010)	1,830 (1,630–2,050)	353 (266–424)	692 (276–798)	30.1 (23.4–34.4)
Type IV-3	1.04	>50	472 (449–495)	1,280 (1,040–1,570)	347 (294–410)	701 (468–781)	102 (99.0–105)	69.8 (60.7–86.4)
Type IV-4	1.09	64	7,700 (4,900–9,600)	371 (321–430)	449 (366–550)	179 (163–191)	113 (70.4–164)	1,430 (985–1,630)

<sup>a</sup> Type I-4 data are from Corsi et al. (34).

**TABLE 3. Concentrations of Alkylphenols, Alkylphenol Ethoxylates, 4-Methyl-1H-benzotriazole, and 5-Methyl-1H-benzotriazole in Five Type I Aircraft Deicers and Four Type IV Aircraft Anti-Icers from Four Different Manufacturers**

	Type I-1a	Type I-1b	Type I-2	Type I-3	Type I-4	Type IV-1	Type IV-2	Type IV-3	Type IV-4
<b>APnEO (ng/g)</b>									
NP	262	<198	<198	<198	<198	<198	<198	<198	1,070
NP1EO	37,000	<204	<204	<204	<204	<204	<204	<204	17,200
NP2EO	25,900	309	<62	<62	640	<62	442	2,940	268,000
NP3EO	11,300	131	<65	<65	1,400	<65	953	7,020	554,000
NP4EO	17,500	308	<61	<61	235	<61	1,330	8,460	581,000
NP5EO	36,100	485	<59	<59	152	<59	2,080	7,850	594,000
NP6EO	70,800	940	<94	<94	<94	<94	4,450	7,190	626,000
NP7EO	85,600	1,120	<114	<114	<114	<114	6,520	4,360	400,000
NP8EO	97,700	1,320	<125	<125	<125	<125	9,160	2,480	235,000
NP9EO	95,400	1,360	<123	<123	<123	<123	11,600	1,230	123,000
NP10EO	97,800	1,170	<110	<110	<110	<110	13,100	644	63,500
NP11EO	71,800	973	<90	<90	<90	<90	13,600	318	25,800
NP12EO	64,700	762	<68	<68	95	<68	13,300	258	12,000
NP13EO	45,400	537	<47	<47	86	<47	11,400	162	3,870
NP14EO	19,600	352	<31	<31	110	<31	9,920	150	1,180
NP15EO	17,100	215	<18	<18	111	<18	6,920	125	395
NP16EO	13,500	130	<11	<11	90	<11	5,460	96	163
OP	<221	<221	<221	<221	<221	<221	<221	<221	<221
OP1EO	338	<216	<216	<216	<216	<216	116,000	<216	<216
OP2EO	<71	<71	<71	<71	260	<71	279,000	<71	<71
OP3EO	<63	<63	<63	<63	289	<63	243,000	<63	<63
OP4EO	<71	<71	<71	<71	522	<71	193,000	153	<71
OP5EO	506	<67	<67	<67	628	<67	140,000	98	<67
other OPnEO	6–15	none	none	none	6–15	none	6–15	6–13	none
ethoxomers detected									
NPnEO mean <i>n</i>	8.6	8.9			4.8		10.8	5.2	5.2
total NPEO	808,000	10,100	BQL <sup>a</sup>	ND	2,920	ND	110,000	43,300	3,510,000
total OPEO	844	ND	ND	BQL	1,700	ND	971,000	251	ND
MeBT (μg/g)									
MeBT-4	196	<100	<100	<100	230	210	782	<100	80
MeBT-5	280	<100	<100	<100	290	252	1,130	<100	100

<sup>a</sup> BQL, below quantification limit.

ethoxomer numbers with the available data since OPnEO ethoxomers were only quantified to *n* = 5.

Concentrations of the NPnEO degradation product NP were above the detection limit (198 ng/g) in formulations

Type I-1a and Type IV-4 but not in other formulations. Octylphenol was not detected in the ADAF formulations.

MeBT was detected in five of the nine formulations with MeBT-5 representing 55–59% of the total MeBT mixture in

TABLE 4. Toxicity Test Results for Selected Additives to Aircraft Deicers and Anti-Icers<sup>a</sup>

ADAF component	acute			chronic		
	Microtox (EC50)	<i>P. promelas</i> (LC50)	<i>C. dubia</i> (LC50)	<i>P. promelas</i> (IC25)	<i>C. dubia</i> (IC25)	<i>S. capricornutum</i> (IC25)
5-MeBT	4.25 (4.18–4.35)	22.0 (20.5–23.5)	81.3 (70.3–95.1)			
4,5-MeBT	6.08 (5.78–6.55)	30.1 (27.3–33.1)	80.7 (67.4–96.6)	21.5 (11.1–24.1)	5.7 (4.6–7.3)	23.8 (13.0–32.8)
NPEO mix	443 (440–445)	3.54 <sup>c</sup>	6.37 (5.71–7.12)			
PG <sup>b</sup>	83,500 (82,000–85,900)	55,800	18,300	6,900	13,500	15,200
EG <sup>b</sup>	133,000 (128,000–137,000)	72,900	34,400	22,500	12,300	5,340

<sup>a</sup> All units are expressed in mg/L. <sup>b</sup> PG and EG data for *P. promelas* and *C. dubia* are from Pillard (10); PG and EG data for *S. capricornutum* are from Pillard and DuFresne (33). <sup>c</sup> 95% confidence interval not reliable because mortality in consecutive dilutions (2.5 and 5.0 mg/L) went from 0 to 100%.

TABLE 5. Relative Toxicity Units (TU<sup>a</sup>) of Aircraft Deicer and Anti-Icer Additive Ingredients as Compared to Toxicity of Five Type I Aircraft Deicing Fluids and Four Type IV Aircraft Anti-Icing Fluids from Four Different Manufacturers

	Type I-1a	Type I-1b	Type I-2	Type I-3	Type I-4	Type IV-1	Type IV-2	Type IV-3	Type IV-4
<b>Microtox</b>									
Glycol (PG or EG) <sup>b</sup>	0.02	0.49	0.06	0.16	0.09	<0.01	0.03	<0.01	0.04
5-MeBT	0.11	ND <sup>d</sup>	ND	ND	0.89	0.04	1.3	ND	0.20
4,5-MeBT	0.13	ND	ND	ND	1.1	0.05	1.5	ND	0.25
NPEO mixture	<0.01	<0.01	ND	ND	<0.01	ND	<0.01	<0.01	0.07
<b><i>P. promelas</i> LC50</b>									
Glycol (PG or EG) <sup>b</sup>	0.03	0.51	0.11	0.20	0.35	0.02	<0.01	0.01	<0.01
5-MeBT	0.03	ND	ND	ND	0.36	0.02	0.05	ND	<0.01
4,5-MeBT	0.03	ND	ND	ND	0.47	0.03	0.06	ND	<0.01
NPEO mixture	0.47	0.09	ND	ND	0.03	ND	0.03	0.02	0.44
<b><i>C. dubia</i> LC50</b>									
Glycol (PG or EG) <sup>b</sup>	0.17	2.2	0.30	0.39	0.47	0.02	0.05	0.01	<0.01
5-MeBT	0.01	ND	ND	ND	0.06	<0.01	0.03	ND	<0.01
4,5-MeBT	0.02	ND	ND	ND	0.11	<0.01	0.05	ND	<0.01
NPEO mixture	0.46	0.08	ND	ND	<0.01	ND	0.03	<0.01	0.29
<b><i>S. capricornutum</i> IC25</b>									
Glycol (PG or EG) <sup>b</sup>	0.02	<0.01	<0.01	0.02	0.87	<0.01	<0.01	<0.01	0.19
5-MeBT	NA <sup>c</sup>	NA	ND	ND	NA	NA	NA	ND	NA
4,5-MeBT	<0.01	ND	ND	ND	0.11	<0.01	<0.01	ND	<0.01
NPEO-9 <sup>e</sup>	0.02	<0.01	ND	ND	<0.01	ND	<0.01	<0.01	0.50

<sup>a</sup> TU = (fraction of additive in ADAF × endpoint concentration for ADAF)/(endpoint concentration for additive). <sup>b</sup> Type I-4 and Type IV-4 are ethylene glycol based. All others are propylene glycol based. <sup>c</sup> NA; Information not available. <sup>d</sup> ND; additive not detected. <sup>e</sup> NPEO-9 IC25 for *S. capricornutum* from previously published literature (17).

each of these five formulations. Formulation Type IV-2 had more than three times the MeBT present than other formulations.

**Additive Contributions to ADAF Toxicity.** *P. promelas* was more sensitive than *C. dubia* in acute tests with three additives; a pattern also seen in four ADAF formulations, all of which contained NPEO, two of which contained OPEO, and two of which contained 4,5-MeBT (Table 4). The Microtox test was more sensitive than *P. promelas* and *C. dubia* to 4- and 5-MeBT, but less sensitive to the NPnEO mixture.

TUs indicated that glycol or selected additives explained toxicity endpoint concentrations for a portion of ADAF formulations for some of the organisms, but each formulation contained other components that were responsible for some observed toxicity (Table 5). In 36 total combinations of test organisms and ADAF formulations (four organisms × nine ADAF formulations), 16 instances indicated that glycol or the selected additives did not greatly influence ADAF toxicity (TU < 0.1), seven instances explained more toxicity with 0.1 < TU < 0.35, nine instances explained more ADAF toxicity yet with 0.35 ≤ TU < 0.55, and only four instances explained most ADAF toxicity with TU > 0.85. NPEO appeared to be

an important factor for toxicity in two of the nine formulations (Type I-1a and Type IV-4), but it was apparent that even these two formulations had other additives contributing to toxicity, since TU values for NPEO were all 0.47 or less. MeBT appeared to be important with regard to Microtox toxicity for two formulations (Type I-4 and Type IV-2) and *P. promelas* toxicity for one formulation (Type I-4), but it was apparent that other unidentified additives contributed toxicity beyond that of MeBT. TU values from Type I formulations indicated that glycol was important with regard to *C. dubia* toxicity for all but one Type I formulation. Glycol was important in Microtox results from formulation Type I-1b primarily because this formulation was relatively nontoxic in the Microtox test. *S. capricornutum* endpoint concentrations were not influenced by additives identified here and were only influenced by glycol in one formulation. Toxicity in formulations Type IV-1 and Type IV-3 was not explained by glycol, NPEO, or MeBT for any of the organisms.

At times, TU values exceeded 1.0 which could be explained either by uncertainty of the various analyses leading into TU computation or by synergistic/antagonistic interactions that changed toxicity of additives when other ADAF components

were present or absent. This was an area of study that was not explored during this research.

While Table 5 is a starting point for explaining the cause of toxicity in ADAF formulations, there is much room for further research to account for the remaining toxicity. To start, OPEO contributions to toxicity were difficult to assess with the available data. First, because the entire distribution was not quantified, and second, because toxicity data for OPEO were not readily available except for the degradation product octylphenol.

Also with regard to APEO surfactants, the information presented in Tables 2–4 does not account for degradation products that could be present in the receiving water with potential to contribute toxicity. NP, OP, smaller chain ethoxomers, and carboxylated alkylphenols are all degradation products of APEOs that may contribute toxicity or endocrine disruption and have potential to accumulate in sediments and tissues (35). In fact, NP, OP, and the smaller chain ethoxomers are more toxic than higher chain ethoxomers (19) which are mostly what is contained in surfactants found in ADAF formulations. Potential ecological effects of other additives and degradation products of other additives have not been studied with regard to ADAF runoff.

Since ADAF additives addressed in this paper account for only some toxicity in the Type I and Type IV formulations tested, discussion of toxicity from other possible additive ingredients is warranted. Alcohol ethoxylates are a class of surfactants that have been identified in ADAF formulations (7). Toxicity results in the literature and from the U.S. EPA Ecotox database indicate that toxicity endpoint concentrations for alcohol ethoxylates are similar to those published for APEO distributions (36, 19) with acute mortality endpoint concentrations typically less than 20 mg/L and chronic endpoint concentrations less than 1 mg/L for some organisms. This suggests that toxicity of alcohol ethoxylates may account for a portion of observed toxicity in ADAF formulations. The primary difference between the APEO surfactants and alcohol ethoxylate surfactants regarding ecological impact is that the degradation products of APEO are thought to be more harmful. However, most research on both classes of surfactants regarding environmental impact revolves around treated effluent rather than direct discharge to the environment. In this respect, ADAF runoff is unique since these surfactants are released directly to the environment without treatment. The parent products will more likely be present in receiving water at much higher concentrations than the degradation products.

There are numerous other additives with parent or degradation products that may or may not (a) be of toxicological significance, (b) have implications as endocrine disruptors, or (c) persist in sediments or tissues. In addition, synergistic and antagonistic effects of the numerous contaminants contained in ADAF formulations as well as the degradation products of these contaminants have yet to be studied. Until the ingredients are either revealed by the manufacturers or identified through other investigations, uncertainties will exist as to the true causes and depths of environmental implications of ADAF runoff, and these contaminants will be released without public knowledge of their environmental impact.

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## Supporting Information Available

NPnEO ethoxomer distribution used for toxicity tests to approximate NPnEO effects in aircraft deicing fluid. This material is available free of charge via the Internet at <http://pubs.acs.org>.

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